WHAT IS CLAIMED IS:

1. An isolated peptide selected from the group consisting of:

(a) a peptide selected from the group consisting of peptides comprising amino acid sequences set forth in SEQ ID NOs:3, 6, 9, 12, 15, 18, 21, 24, 27, 30, 33, 36, 39, 42, 45, 48, 51, 54, 57, 60, 63, 66, 69, 72, 75, 78, 81, 84, 87, 90, 93, 96, 99, 102, 105, 108, 111, 114, 117, 120, 123, 126, 129, 132, 135, 138, 141, 144, 147, 150, 153, 156, 159, 162, 165, 168, 171, 174, 177, 180, 183, 186, 189, 192, 195, 198, 201, 204, 207, 210, 216, 219, 222, 225, 228-287, 289-315, 319-321, 323-337 and 340; and

(b) analogs and derivatives of the peptide in (a).

2. The peptide of claim 1, wherein Xaa1 is Glu or γ-carboxy-Glu, Xaa2 is Gln or pyro-Glu, Xaa3 is Pro or *trans*-4-hydroxy-Pro, Xaa4 is D or L Trp or D or L 6-bromo-Trp, and Xaa5 is Tyr, mono-iodo-Tyr, ^{125}I –Tyr, di-iodo-Tyr, O-sulpho-Tyr or O-phospho-Tyr.

3. The peptide derivative of claim 1, in which Arg residues may be substituted by Lys, ornithine, homoarginine, *nor*-Lys, N-methyl-Lys, N,N-dimethyl-Lys, N,N,N-trimethyl-Lys or any synthetic basic amino acid; the Lys residues may be substituted by Arg, ornithine, homoarginine, *nor*-Lys, or any synthetic basic amino acid; the Tyr residues may be substituted with *meta*-Tyr, *ortho*-Tyr, *nor*-Tyr, ^{125}I-Tyr, mono-halo-Tyr, di-halo-Tyr, O-sulpho-Tyr, O-phospho-Tyr, nitro-Tyr or any synthetic hydroxy containing amino acid; the Ser residues may be substituted with Thr or any synthetic hydroxylated amino acid; the Thr residues may be substituted with Ser or any synthetic hydroxylated amino acid; the Phe residues may be D or L, may be substituted at the *ortho*, *meta*, and/or *para* positions with a halogen or may be substituted with any synthetic aromatic amino acid; the Trp residues may be substituted with Trp (D), *neo*-Trp, 6-halo-Trp (D or L), preferably 6-halo, or any aromatic synthetic amino acid; the Asn, Ser, Thr or Hyp residues may be substituted with a glycan; the halogen may be iodo, chloro, fluoro or bromo; the Tyr residues may also be substituted with the 3-hydroxyl or 2-hydroxyl isomers (meta-Tyr or ortho-Tyr, respectively) and corresponding O-sulpho- and O-phospho-derivatives; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g., tetrazolyl derivatives of Gly and Ala; the Leu may be

substituted with Leu (D); the Glu residues may be substituted with Gla or Asp; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g. tetrazolyl derivatives of Gly and Ala; the N-terminal Gln may be substituted with pyro-glutamate (Z); the aliphatic amino acids may be substituted by synthetic derivatives bearing non-natural aliphatic branched or linear side chains C_nH_{2n+2} up to and including n=8; the Met residues may be substituted with *nor*-leucine (Nle); the Cys residues may be in D or L configuration and may optionally be substituted with homocysteine (D or L); basic residues in the backbone may be D or L configuration; the central Trp residue within the beta-turn is preferably epimerized to the D-form; pairs of Cys residues may be replaced pairwise with isoteric lactam or ester-thioether replacements, such as Ser/(Glu or Asp), Lys/(Glu or Asp), Cys/(Glu or Asp) or Cys/Ala combinations; and individual Cys residues may be replaced with homoCys, seleno-Cys or penicillamine, so that disulfide bridges may be formed between Cys-homoCys or Cys-penicillamine, or homoCys-penicllamine.

4. The peptide derivative of claim 3, wherein the glycan is any N-, S- or O-linked mono-, di-, tri-, poly- or oligosaccharide that can be attached to any hydroxy, amino or thiol group of natural or modified amino, wherein the monosaccharides making up the glycan can be unmodified or modified D-allose, D-altrose, D-glucose, D-mannose, D-gulose, D-idose, D-galactose, D-talose, D-galactosamine, D-glucosamine, D-N-acetyl-glucosamine (GlcNAc), D-N-acetyl-galactosamine (GalNAc), D-fucose or D-arabinose, wherein the glycosidic linkage is beta and 1-4 or 1-3, preferably 1-3, and wherein the linkage between the glycan and the amino acid may be alpha or beta, preferably alpha and is 1-.

5. The peptide derivative of claim 4, wherein the modification may include one or more O-sulfate, O-phosphate, O-acetyl or acidic groups, and combinations thereof.

6. The peptide derivative of claim 4, wherein the gylcan may also include similar polyhydroxy groups, such as D-penicillamine 2,5 and halogenated derivatives thereof or polypropylene glycol derivatives.

7. The derivative of the peptide of claim 1 in which the peptide is truncated.

8. The peptide derivative of claim 3 in which the peptide derivative is truncated.

9. The peptide derivative of claim 4 in which the peptide derivative is truncated.

10. An peptide of claim 1 containing 4 cysteines which are bridged as [1,4 / 2,3].

11. An peptide of claim 1 containing 4 cysteines which are bridged as [1,3 / 2,4].

12. The peptide of claim 1, wherein the peptide is tagged with a radiolabel.

13. The peptide derivative of claim 1 in which a basic or aromatic amino acid in the beta turn is a D-isomer.

14. The peptide derivative of claim 13 in which the peptide derivative is truncated.

15. The peptide of claim 13, wherein the peptide is tagged with a radiolabel.

16. The peptide of claim 14, wherein the peptide is tagged with a radiolabel.

17. An isolated nucleic acid encoding a β-superfamily conopeptide propeptide selected from the group of propeptides comprising amino acid sequences set forth in SEQ ID NO:2, 5, 8, 11, 14, 17, 20, 23, 26, 29, 32, 35, 38, 41, 44, 47, 50, 53, 56, 59, 62, 65, 68, 71, 74, 77, 80, 83, 86, 89, 92, 95, 98, 101, 104, 107, 110, 113, 116, 119, 122, 125, 128, 131, 134, 137, 140, 143, 146, 149, 152, 155, 158, 161, 164, 167, 170, 173, 176, 179, 182, 185, 188, 191, 194, 197, 200, 203, 206, 209, 215, 218, 221, 224, and 227.

18. The isolated nucleic acid of claim 17 wherein the nucleic acid comprises a nucleotide sequence selected from the group of nucleotide sequences set forth in SEQ ID NO:1, 4, 7, 10, 13, 16, 19, 22, 25, 28, 31, 34, 37, 40, 43, 46, 49, 52, 55, 58, 61, 64, 67, 70, 73, 76, 79, 82, 85, 88, 91, 94, 97, 100, 103, 106, 109, 112, 115, 118, 121, 124, 127, 130, 133,

136, 139, 142, 145, 148, 151, 154, 157, 160, 163, 166, 169, 172, 175, 178, 181, 184, 187, 190, 193, 196, 199, 202, 205, 208, 214, 217, 220, 223, and 226.

19. An isolated β-superfamily conopeptide propeptide selected from the group of propeptides comprising amino acid sequences set forth in SEQ ID NO:2, 5, 8, 11, 14, 17, 20, 23, 26, 29, 32, 35, 38, 41, 44, 47, 50, 53, 56, 59, 62, 65, 68, 71, 74, 77, 80, 83, 86, 89, 92, 95, 98, 101, 104, 107, 110, 113, 116, 119, 122, 125, 128, 131, 134, 137, 140, 143, 146, 149, 152, 155, 158, 161, 164, 167, 170, 173, 176, 179, 182, 185, 188, 191, 194, 197, 200, 203, 206, 209, 215, 218, 221, 224, and 227.

20. A method for treating cancer which comprises administering an active agent or a pharmaceutically acceptable salt thereof to an individual having cancer, wherein said active agent is a peptide tagged with a radionuclide, wherein said peptide is a β-superfamily conotoxin.

21. The method of claim 20, wherein said β-superfamily conotoxin is selected from the group consisting of:

(a) a peptide selected from the group consisting of peptides comprising amino acid sequences set forth in SEQ ID NO:3, 6, 9, 12, 15, 18, 21, 24, 27, 30, 33, 36, 39, 42, 45, 48, 51, 54, 57, 60, 63, 66, 69, 72, 75, 78, 81, 84, 87, 90, 93, 96, 99, 102, 105, 108, 111, 114, 117, 120, 123, 126, 129, 132, 135, 138, 141, 144, 147, 150, 153, 156, 159, 162, 165, 168, 171, 174, 177, 180, 183, 186, 189, 192, 195, 198, 201, 204, 207, 210, 216, 219, 222, 225, 228-287, 289-315, 319-321, 323-337 and 340; and

(b) analogs and derivatives of the peptide in (a).

22. The method of claim 20, wherein the radionuclide is selected from the group consisting of ^{131}iodine, ^{123}iodine, ^{99m}technicium, ^{111}indium, ^{188}rhenium, ^{186}rhenium, ^{67}gallium, ^{90}yttrium, ^{105}rhodium, ^{89}strontium, ^{153}samarium, ^{211}astatine, ^{212}bismuth, ^{213}bismuth, ^{177}lutetium, ^{64}copper, ^{67}copper, ^{47}scandium, ^{109}palladium.

23. The method of claim 21, wherein the radionuclide is selected from the group consisting of ^{131}iodine, ^{123}iodine, ^{99m}technicium, ^{111}indium, ^{188}rhenium, ^{186}rhenium, ^{67}gallium,

^{90}yttrium, ^{105}rhodium, ^{89}strontium, ^{153}samarium, ^{211}astatine, ^{212}bismuth, ^{213}bismuth, ^{177}lutetium, ^{64}copper, ^{67}copper, ^{47}scandium, ^{109}palladium.

24. The method of claim 21, wherein the active agent a peptide derivative in which Arg residues may be substituted by Lys, ornithine, homoarginine, *nor*-Lys, N-methyl-Lys, N,N-dimethyl-Lys, N,N,N-trimethyl-Lys or any synthetic basic amino acid; the Lys residues may be substituted by Arg, ornithine, homoarginine, *nor*-Lys, or any synthetic basic amino acid; the Tyr residues may be substituted with *meta*-Tyr, *ortho*-Tyr, *nor*-Tyr, ^{125}I-Tyr, mono-halo-Tyr, di-halo-Tyr, O-sulpho-Tyr, O-phospho-Tyr, nitro-Tyr or any synthetic hydroxy containing amino acid; the Ser residues may be substituted with Thr or any synthetic hydroxylated amino acid; the Thr residues may be substituted with Ser or any synthetic hydroxylated amino acid; the Phe residues may be D or L, may be substituted at the *ortho*, *meta*, and/or *para* positions with a halogen or may be substituted with any synthetic aromatic amino acid; the Trp residues may be substituted with Trp (D), *neo*-Trp, 6-halo-Trp (D or L), preferably 6-halo, or any aromatic synthetic amino acid; the Asn, Ser, Thr or Hyp residues may be substituted with a glycan; the halogen may be iodo, chloro, fluoro or bromo; the Tyr residues may also be substituted with the 3-hydroxyl or 2-hydroxyl isomers (meta-Tyr or ortho-Tyr, respectively) and corresponding O-sulpho- and O-phospho-derivatives; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g., tetrazolyl derivatives of Gly and Ala; the Leu may be substituted with Leu (D); the Glu residues may be substituted with Gla or Asp; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g. tetrazolyl derivatives of Gly and Ala; the N-terminal Gln may be substituted with pyro-glutamate (Z); the aliphatic amino acids may be substituted by synthetic derivatives bearing non-natural aliphatic branched or linear side chains C_nH_{2n+2} up to and including n=8; the Met residues may be substituted with *nor*-leucine (Nle); the Cys residues may be in D or L configuration and may optionally be substituted with homocysteine (D or L); basic residues in the backbone may be D or L configuration; the central Trp residue within the beta-turn is preferably epimerized to the D-form; pairs of Cys residues may be replaced pairwise with isoteric lactam or ester-thioether replacements, such as Ser/(Glu or Asp), Lys/(Glu or Asp), Cys/(Glu or Asp) or Cys/Ala combinations; and individual Cys residues may be replaced with homoCys, seleno-Cys

or penicillamine, so that disulfide bridges may be formed between Cys-homoCys or Cys-penicillamine, or homoCys-penicllamine.

25. The method of claim 24, wherein the the glycan is any N-, S- or O-linked mono-, di-, tri-, poly- or oligosaccharide that can be attached to any hydroxy, amino or thiol group of natural or modified amino, wherein the monosaccharides making up the glycan can be unmodified or modified D-allose, D-altrose, D-glucose, D-mannose, D-gulose, D-idose, D-galactose, D-talose, D-galactosamine, D-glucosamine, D-N-acetyl-glucosamine (GlcNAc), D-N-acetyl-galactosamine (GalNAc), D-fucose or D-arabinose, wherein the glycosidic linkage is beta and 1-4 or 1-3, preferably 1-3, and wherein the linkage between the glycan and the amino acid may be alpha or beta, preferably alpha and is 1-.

26. The method of claim 21, wherein the peptide has activity at somatostatin receptors.

27. A method of alleviating pain in an individual which comprises administering to an individual who is either exhibiting pain or is about to be subjected to a pain-causing event a pain-alleviating amount of an active agent or a pharmaceutically acceptible salt thereof, wherein said active agent is a β-superfamily conotoxin.

28. The method of claim 27, wherein the βsuperfamily conotoxin is selected from the group consisting of:

(a) a peptide selected from the group consisting of peptides comprising amino acid sequences set forth in SEQ ID NO:3, 6, 9, 12, 15, 18, 21, 24, 27, 30, 33, 36, 39, 42, 45, 48, 51, 54, 57, 60, 63, 66, 69, 72, 75, 78, 81, 84, 87, 90, 93, 96, 99, 102, 105, 108, 111, 114, 117, 120, 123, 126, 129, 132, 135, 138, 141, 144, 147, 150, 153, 156, 159, 162, 165, 168, 171, 174, 177, 180, 183, 186, 189, 192, 195, 198, 201, 204, 207, 210, 216, 219, 222, 225, 228-287, 289-315, 319-321, 323-337 and 340; and

(b) analogs and derivatives of the peptide in (a).

29. The method of claim 28, wherein the active agent a peptide derivative in which Arg residues may be substituted by Lys, ornithine, homoarginine, *nor*-Lys, N-methyl-Lys, N,N-dimethyl-Lys, N,N,N-trimethyl-Lys or any synthetic basic amino acid; the Lys

residues may be substituted by Arg, ornithine, homoarginine, *nor*-Lys, or any synthetic basic amino acid; the Tyr residues may be substituted with *meta*-Tyr, *ortho*-Tyr, *nor*-Tyr, ^{125}I-Tyr, mono-halo-Tyr, di-halo-Tyr, O-sulpho-Tyr, O-phospho-Tyr, nitro-Tyr or any synthetic hydroxy containing amino acid; the Ser residues may be substituted with Thr or any synthetic hydroxylated amino acid; the Thr residues may be substituted with Ser or any synthetic hydroxylated amino acid; the Phe residues may be D or L, may be substituted at the *ortho*, *meta*, and/or *para* positions with a halogen or may be substituted with any synthetic aromatic amino acid; the Trp residues may be substituted with Trp (D), *neo*-Trp, 6-halo-Trp (D or L), preferably 6-halo, or any aromatic synthetic amino acid; the Asn, Ser, Thr or Hyp residues may be substituted with a glycan; the halogen may be iodo, chloro, fluoro or bromo; the Tyr residues may also be substituted with the 3-hydroxyl or 2-hydroxyl isomers (meta-Tyr or ortho-Tyr, respectively) and corresponding O-sulpho- and O-phospho-derivatives; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g., tetrazolyl derivatives of Gly and Ala; the Leu may be substituted with Leu (D); the Glu residues may be substituted with Gla or Asp; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g. tetrazolyl derivatives of Gly and Ala; the N-terminal Gln may be substituted with pyro-glutamate (Z); the aliphatic amino acids may be substituted by synthetic derivatives bearing non-natural aliphatic branched or linear side chains C_nH_{2n+2} up to and including n=8; the Met residues may be substituted with *nor*-leucine (Nle); the Cys residues may be in D or L configuration and may optionally be substituted with homocysteine (D or L); basic residues in the backbone may be D or L configuration; the central Trp residue within the beta-turn is preferably epimerized to the D-form; pairs of Cys residues may be replaced pairwise with isoteric lactam or ester-thioether replacements, such as Ser/(Glu or Asp), Lys/(Glu or Asp), Cys/(Glu or Asp) or Cys/Ala combinations; and individual Cys residues may be replaced with homoCys, seleno-Cys or penicillamine, so that disulfide bridges may be formed between Cys-homoCys or Cys-penicillamine, or homoCys-penicllamine.

30. The method of claim 29, wherein the the glycan is any N-, S- or O-linked mono-, di-, tri-, poly- or oligosaccharide that can be attached to any hydroxy, amino or thiol group of natural or modified amino, wherein the monosaccharides making up the glycan can be

unmodified or modified D-allose, D-altrose, D-glucose, D-mannose, D-gulose, D-idose, D-galactose, D-talose, D-galactosamine, D-glucosamine, D-N-acetyl-glucosamine (GlcNAc), D-N-acetyl-galactosamine (GalNAc), D-fucose or D-arabinose, wherein the glycosidic linkage is beta and 1-4 or 1-3, preferably 1-3, and wherein the linkage between the glycan and the amino acid may be alpha or beta, preferably alpha and is 1-.

31. The method of claim 27, wherein the pain is visceral pain.

32. A method for treating or preventing disorders associated with a disorder selected from the group consisting of voltage-gated ion channel disorders, ligand-gated ion channel disorders and receptor disorders, such as disorders of G-protein coupled receptors, in an individual which comprises administering to an individual in need thereof a therapeutically effective amount of an active agent or a pharmaceutically acceptible salt thereof, wherein the active agent is a β-superfamily conotoxin.

33. The method of claim 32, wherein the β-superfamily conotoxin is selected from the group consisting of:

(a) a peptide selected from the group consisting of peptides comprising amino acid sequences set forth in SEQ ID NO:3, 6, 9, 12, 15, 18, 21, 24, 27, 30, 33, 36, 39, 42, 45, 48, 51, 54, 57, 60, 63, 66, 69, 72, 75, 78, 81, 84, 87, 90, 93, 96, 99, 102, 105, 108, 111, 114, 117, 120, 123, 126, 129, 132, 135, 138, 141, 144, 147, 150, 153, 156, 159, 162, 165, 168, 171, 174, 177, 180, 183, 186, 189, 192, 195, 198, 201, 204, 207, 210, 216, 219, 222, 225, 228-287, 289-315, 319-321, 323-337 and 340; and

(b) analogs and derivatives of the peptide in (a).

34. The method of claim 33, wherein the active agent a peptide derivative in which Arg residues may be substituted by Lys, ornithine, homoarginine, *nor*-Lys, N-methyl-Lys, N,N-dimethyl-Lys, N,N,N-trimethyl-Lys or any synthetic basic amino acid; the Lys residues may be substituted by Arg, ornithine, homoarginine, *nor*-Lys, or any synthetic basic amino acid; the Tyr residues may be substituted with *meta*-Tyr, *ortho*-Tyr, *nor*-Tyr, ^{125}I-Tyr, mono-halo-Tyr, di-halo-Tyr, O-sulpho-Tyr, O-phospho-Tyr, nitro-Tyr or any synthetic hydroxy containing amino acid; the Ser residues may be substituted with Thr or

any synthetic hydroxylated amino acid; the Thr residues may be substituted with Ser or any synthetic hydroxylated amino acid; the Phe residues may be D or L, may be substituted at the *ortho*, *meta*, and/or *para* positions with a halogen or may be substituted with any synthetic aromatic amino acid; the Trp residues may be substituted with Trp (D), *neo*-Trp, 6-halo-Trp (D or L), preferably 6-halo, or any aromatic synthetic amino acid; the Asn, Ser, Thr or Hyp residues may be substituted with a glycan; the halogen may be iodo, chloro, fluoro or bromo; the Tyr residues may also be substituted with the 3-hydroxyl or 2-hydroxyl isomers (meta-Tyr or ortho-Tyr, respectively) and corresponding O-sulpho- and O-phospho-derivatives; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g., tetrazolyl derivatives of Gly and Ala; the Leu may be substituted with Leu (D); the Glu residues may be substituted with Gla or Asp; the acidic amino acid residues may be substituted with any synthetic acidic amino acid, e.g. tetrazolyl derivatives of Gly and Ala; the N-terminal Gln may be substituted with pyro-glutamate (Z); the aliphatic amino acids may be substituted by synthetic derivatives bearing non-natural aliphatic branched or linear side chains C_nH_{2n+2} up to and including n=8; the Met residues may be substituted with *nor*-leucine (Nle); the Cys residues may be in D or L configuration and may optionally be substituted with homocysteine (D or L); basic residues in the backbone may be D or L configuration; the central Trp residue within the beta-turn is preferably epimerized to the D-form; pairs of Cys residues may be replaced pairwise with isoteric lactam or ester-thioether replacements, such as Ser/(Glu or Asp), Lys/(Glu or Asp), Cys/(Glu or Asp) or Cys/Ala combinations; and individual Cys residues may be replaced with homoCys, seleno-Cys or penicillamine, so that disulfide bridges may be formed between Cys-homoCys or Cys-penicillamine, or homoCys-penicllamine.

35. The method of claim 34, wherein the the glycan is any N-, S- or O-linked mono-, di-, tri-, poly- or oligosaccharide that can be attached to any hydroxy, amino or thiol group of natural or modified amino, wherein the monosaccharides making up the glycan can be unmodified or modified D-allose, D-altrose, D-glucose, D-mannose, D-gulose, D-idose, D-galactose, D-talose, D-galactosamine, D-glucosamine, D-N-acetyl-glucosamine (GlcNAc), D-N-acetyl-galactosamine (GalNAc), D-fucose or D-arabinose, wherein the

glycosidic linkage is beta and 1-4 or 1-3, preferably 1-3, and wherein the linkage between the glycan and the amino acid may be alpha or beta, preferably alpha and is 1-.

36. The method of claim 32, wherein the disorder is a G-protein coupled receptor disorder.

37. The method of claim 36, wherein the G-protein coupled receptor is selected from the group consisting of sst, cortistatin (CST), melanocortin (MC_xR, wherein x = 1, 2, 3, 4, 5), opioid (μ, δ, κ), neurokinin, bradykinin, galanin, CCK_A, CCK_B, endothelin, serotonin, adrenergic receptors, angiotensin, neuropeptide-Y, sigma1, sigma2, oxytocin, CGRP, GRPR, histamine, imidazoline, neurotensin, VIP, vasopressin, substance K, chemokine receptors, CRF_1, CRF_{2a}, $CRF_{2\beta}$, $CRF_{2\gamma}$, CRF-BP, orexin, urotensin, glycoprotein IIb/IIIa, thrombin receptors and orphan GPCRs.

38. The method of claim 36, wherein the GPCR is selected from the group consisting of MCH_2R/SLT, SP1999/P_2Y_{12}, $CRTH_2$, $NPFF_1$, $NPFF_2$, HH_4R, h-GPR_{54}, $CysLT_2$, neuromedin receptors, $BLTR_2$, G_2A, TA_1, LTB_4, ghrelin, motilin MTL-R, purinergic receptors, muscarinic receptors, ORL-1, apelin, CB_1, CB_2 and GPCRs of orphan status having no known cognate ligand.

39. The method of claim 32, wherein the disorder is selected from the group consisting of cancer, neoplasm, solid tumor, diabetic nephropathy, fibrosis, hypophysis tumor, GI disease, IBS, restinosis, angiogenesis disorder, diabetes mellitus, endocrine tumor, diarrhea, pancreatic disease, prostate tumor, bleeding and apoptosis.

40. The method of claim 39, wherein the peptide has activity at somatostatin receptors.

41. The method of claim 32, wherein the disorder is selected from the group consisting of inflammation, pain, diabetes, obesity, sexual dysfunction, acromegaly, glaucoma, cardiovascular, diabetic, retinopathy, depression, myocardial infarction, stroke, epilepsy, anorexia, wasting diseases, seborrheic dermatitis, schizophrenia, mood disorders, chemotherapeutic induced emesis, disorders associated with changes in blood pressure,

immune disorders, nerve damage, acne, GI infections, myocardial infarction, angina, thromboembolism and cardiovascular disease.

42. The method of claim 41, wherein the peptide has activity at somatostatin receptors or at melanocortin receptors.

43. The method of claim 32, wherein the receptor is LHRH.

44. The method of claim 43, wherein the disorder is osteoporosis.

45. The method of claim 32, wherein the disorder is associated with a melanocortin system or MCR dysfunction.

46. The method of claim 45, wherein the disorder is selected from the group consisting of erectile dysfunction, obesity inflammation and melanoma.

47. The method of claim 45, wherein the peptide contains a β-turn.

48. The method of claim 47, wherein the peptide is a β-Ge14.1 analogue, derivative thereof or pharmaceutically acceptable salt thereof, wherein the β-Ge14.1 analogue is selected from the group of peptides comprising an amino acid sequence set forth in SEQ ID NOs:334-337,.

49. A method for identifying drug candidates for use as treating or preventing disorders associated with a disorder selected from the group consisting of voltage-gated ion channel disorders, ligand-gated ion channel disorders and receptor disorders, such as disorders of G-protein coupled receptors which comprises screening a drug candidate for its action at or partially at the same functional site as a β-superfamily conotoxin and capable of elucidation of similar functional response as said conotoxin.

50. The method of claim 49, wherein the displacement of a labeled β-superfamily conotoxin from its receptor or other complex by a candidate drug agent is used to identify suitable candidate drugs.

51. The method of claim 49, wherein a biological assay on a test compound to determine the therapeutic activity is conducted and compared to the results obtained from the biological assay of a β-superfamily conotoxin.

52. The method of claim 49, wherein the binding affinity of a small molecule to the receptor of a β-conotoxin is measured and compared to the binding affinity of a β-superfamily conotoxin to its receptor.

53. A method of identifying compounds that mimic the therapeutic activity of a β-superfamily conotoxin, comprising the steps of: (a) conducting a biological assay on a test compound to determine the therapeutic activity; and (b) comparing the results obtained from the biological assay of the test compound to the results obtained from the biological assay of a β-superfamily conotoxin.

54. A method for characterizing a new site on a voltage-gated ion channel, a ligand-gated ion channel and a receptor, such as a G-protein coupled receptorswhich comprises contacting a peptide of claim 1 with a channel or receptor and measuring the binding of the peptide with the channel or receptor or by measuring a functional *in vitro* parameters such as fluorescence, phosphorescence and illuminescence..

55. The method of claim 54, wherein the receptor is a G-protein coupled receptor.

56. The method of claim 54, wherein the peptide is radiolabeled.

57. A method for designing a β-beta turn mimetic of a β-superfamily conotoxin containing a β-turn motif selected from the group consistinf of (i) a –CX1X2KX1C- (SEQ ID NO:338) motif, wherein X1 is any amino acid and X2 is D or L Trp or D or L 6-bromo Trp and (ii) a –CPX3RVC- (SEQ ID NO:339) motif, wherein X3 is D or L Phe, which

comprises replacing this motif with a non-peptide turn mimetic β-turn scaffold and then attaching receptor binding domains contained within the N and C-terminal sequences of a β-superfamily conotoxin to the β-turn scaffold to mimic the 3D spatial array within the native β-superfamily conotoxin.

58. A method for identifying a ligand which binds to an orphan G-protein coupled receptor (orphan GPCR) which comprises contacting a peptide of claim 1 or a radiolabeled derivative of the peptide with an orphan GPCR and measuring the amount of binding of the peptide to the orphan GPCR.

59. The method of claim 58, wherein the peptide is radiolabeled.

60. The method of claim 59, wherein the radiolabel is selected from the group consisting of ^{3}H and ^{125}I.

61. The method of claim 58, which further comprises performing a homology search for the peptide which binds to the orphan GPCR to identify other candidate ligands for testing.